no suppressibility with dexamethasone and no response to metyrapone are expected. The interpretation of such tests should, however, take into account—in addition to the intra-assay and interassay coefficient of variation for the analytical determinations—that the sensitivity and specificity for Cushing's disease of both these tests are less than 100%. As shown in Table 1, among patients with the ectopic ACTH syndrome, 48% show an apparent response to metyrapone and 20% to dexamethasone.

To achieve a better separation of pituitary versus ectopic sources of ACTH, some authors advocate the use of higher doses of dexamethasone (32 mg) in patients who fail to respond to the standard 8-mg test.[1-3] We warn about using such a maneuver, which is based on a single case report,[4] has not been systematically evaluated and may, as in the case of our patient, lead to an erroneous diagnosis. The availability of CRF, the hypothalamic polypeptide that modulates pituitary ACTH secretion, provides a novel tool for the differential diagnosis of Cushing's syndrome.[5] Review (Table 1) of the 49 published cases of Cushing's disease tested with CRF shows that 46 (94%) patients showed a response while only 3 (6%) patients showed no response and, of the 14 patients with ectopic ACTH secretion, 13 showed no response to CRF and 1 patient showed some responsiveness that was, however, not reproducible. If further data confirm this trend, the CRF test could become very useful in the etiologic diagnosis of Cushing's syndrome and would have a greater sensitivity and specificity than the standard 8-mg dexamethasone test. The addition of this simple test to the diagnostic evaluation could in some patients reduce the need for more invasive and expensive diagnostic procedures such as inferior petrosal sinus catheterization.

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Acquired Immunodeficiency Syndrome Presenting as Schizophrenia

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SEVERAL INVESTIGATORS have reported affective and psychotic symptoms with the acquired immunodeficiency syndrome (AIDS). Nurnberg and co-workers reported the case of a 28-year-old man with AIDS who presented with command hallucinations, persecutory delusions, anorexia, a 13.6-kg weight loss (30 lb), anhedonia, abulia, middle insomnia, psychomotor retardation and impaired recent memory.[1] Hoffman reviewed two AIDS cases that had in common a psychiatric presentation and diffuse cerebral atrophy indicated by computed tomography.[2] One case was characterized as progressive dementia and the other as transient acute encephalopathy. Kermani and associates identified a triad of mood disturbance, thought disorder with grandiose delusions and severe memory deficits in a series of three patients with AIDS.[3] Subsequently, Kermani and colleagues described the case of an AIDS patient who presented with manic features before development of cognitive and memory impairment,[4] while Thomas and co-workers reported seeing a 22-year-old patient with AIDS who had a paranoid psychosis but normal cognitive and memory functions.[5]

We report a case further illustrating the protean presentation of psychiatric signs and symptoms in AIDS patients and define one pathway—a natural history culminating in dementia. The case is remarkable because of a prolonged course that began with subtle organicity, was punctuated by psychotic decompensation and progressed to profound dementia.

Report of a Case

The patient, a 32-year-old single, bisexual man, was brought to the psychiatric emergency service by his sister. She stated that for the past nine months he had increasing apathy, social withdrawal to the point of almost total isolation and bizarre behavior such as urinating in public. The initial

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ABBREVIATIONS USED IN TEXT

AIDS = acquired immunodeficiency syndrome
CMV = cytomegalovirus
CNS = central nervous system
HIV = human immunodeficiency virus

interview, using the patient and his sister as informants, revealed that he had a 12-month history of a colitis partially responsive to treatment with sulfasalazine (Azulfidine), which shortly preceded the onset of apparent apathy, withdrawal and unusual behavior. Six months before evaluation he had received a short course of amitriptyline hydrochloride (Elavil) for a depressed mood attributed to pain and diarrhea from colitis and to his dissatisfaction with his work. His mood improved. Three months before evaluation he lost his job as a computer data processor because of emotional lability, decreased concentration and diminished productivity. He returned home to live with his parents, who noted his inattentiveness, peculiar behavior and bizarre grimacing. He became increasingly agitated and confided with his married sister that he feared he was losing his mind. During their parents' vacation, the sister became alarmed by his more apparent agitation and eccentric behavior and brought him to the emergency department for evaluation.

The patient said he had no history of behavioral or psychiatric problems before the onset of the current illness. This was corroborated by family members, as was the negative family history for psychiatric disorder.

On mental state examination he appeared disheveled but well developed and was agitated and distracted. He exhibited bizarre facial grimacing. His rate of speech was normal. His thinking was impoverished, his associations were moderately loosened and he manifested occasional blocking. His affect was guarded and he smiled or laughed frequently, which was incongruous with verbal content. He said he did not have hallucinations, but he appeared to look at and listen to internally generated stimuli. He admitted to receiving special messages from television programs and music and felt that the family television could cause his thoughts to vanish. He also believed that his intestine was an independent organism that crawled from side to side "like a worm." Suicidal and homicidal ideas were absent.

He was alert and oriented to person, place and time and could follow simple commands. His forward-digit memory span was seven, and he was able to recall three/three objects after five minutes. He named past presidents as Reagan, Carter, Kennedy, Nixon and Truman. He made several subtraction errors on serial sevens. Proverbs were bizarrely interpreted. To "a rolling stone gathers no moss," he responded "let's canoe down the river." "Even monkeys fall out of trees" was interpreted as "cats do, too."

On the basis of a history of bizarre behavior and steady deterioration of functioning, plus a mental state showing psychotic features and disturbance in attention attributed to agitation and thought disorder, a clinical diagnosis of schizophrenia was made in the psychiatric emergency clinic and the patient was admitted to the psychiatry service.

On admission to the psychiatry unit, the findings of a physical examination were significant for a temperature of 37.9°C (100.2°F), cervical and inguinal lymphadenopathy, diffuse rales and rhonchi, diffuse abdominal tenderness without rebound and multiple hyperpigmented, arcuate skin lesions over the upper back and arms consistent with Kaposi's sarcoma. These findings led to the presumptive diagnosis of AIDS, and the patient was transferred to the medical service.

Laboratory evaluation showed normocytic anemia, relative leukopenia in a patient with infection (leukocyte count 4,200 to 6,600 per μl) and a normal differential without a left shift. His serum lactic dehydrogenase level was elevated at 184 to 288 units per liter, and his γ-glutamyl transferase level was elevated at 75 to 196 IU per liter. Oropharynx cultures were positive for *Candida albicans* and *Candida krusei*, bronchial washings were positive for *Hemophilus influenzae* and fecal cultures were positive for *Cryptospiridium* and *Campylobacter* organisms. Biopsy of skin lesions yielded a diagnosis of Kaposi's sarcoma. A traumatic lumbar puncture showed a total cerebrospinal fluid protein content of 76 mg per dl, glucose 44 mg per dl and 3 lymphocytes per μl. Cultures of cerebrospinal fluid specimens were negative for bacteria, results of serology were normal, and cultures for cytomegalovirus (CMV) and antibody titers for *Toxoplasma gondii* were negative. Chest x-ray films were normal. Urine toxicologic tests for hallucinogens, psychostimulants, sedative-hypnotics, narcotics, alcohol and salicylates were negative.

To document the psychiatric presentation more reliably, a psychiatrist trained in administering the National Institute of Mental Health Diagnostic Interview Schedule (DIS, Version III-A)[6] interviewed the patient two days after his transfer. Based on the interview and initial presentation, the patient satisfied criteria for schizophrenia according to the American Psychiatric Association's *Diagnostic and Statistical Manual of Mental Disorders*, third edition.

By three weeks after admission, a thorough neuroradiologic and neuropsychological examination was completed. In the first week, clinical reassessment showed more apparent cognitive deficits. A computed tomographic scan of the head showed lateral and third ventricular enlargement consistent with early hydrocephalus. Nuclear magnetic resonance imaging of the head was interpreted as normal.

Neuropsychological assessment confirmed cognitive dysfunction. The patient achieved scaled scores of 8 on the Wechsler Adult Intelligence Scale-Revised digit-span and vocabulary subtests; expected scaled scores given the patient's age and education would be 11 or 12 for each subtest.[7] On the subtest Symbol Digit Paired Associate Learning,[8] the patient scored 7 of 28 correct over four trials; testing of control subjects in our laboratory indicates a normal range of 21 to 28 correct items. On Halstead's Category Test the patient made 95 errors per 208 stimuli. The average score for persons of this patient's age and education is 39, and a score above 50 shows definite impairment.[9,10] Wechsler Memory Scale Visual Reproduction Testing showed profound deficits and delay in completing visual motor tasks.[11] The patient was unable to do trail-making tests and Paced Auditory Addition tests. Overall, neuropsychological testing indicated severe deficits in memory, abstracting ability and visual-motor performance.

The patient's psychiatric clinical presentation had evolved from schizophrenia-like symptoms to a more apparent dementia. Over a four-week period, his speech became less productive and more disorganized, his mood changed from silly to sullen with frequent outbursts of hostility. Ideas of reference and delusional material decreased and he later denied having them. On repeat examination he no longer met

criteria for a current diagnosis of schizophrenia and said he did not have the criterion items for a lifetime diagnosis of this disorder. Deficits in memory and orientation persisted. His agitation diminished after treatment with haloperidol (Haldol), 2 to 4 mg given daily by mouth, and the medication was discontinued before discharge without worsening of symptoms. He said he had no interest in his diagnosis or prognosis, and he ceased confiding in his sister. At first, his parents refused his discharge to home and insisted on institutional care. Information about the low infectivity of human immunodeficiency virus (HIV) and an introduction to support services reassured his parents and they accepted his care. His discharge diagnosis was HIV encephalitis.

He was admitted to hospital four times during the remaining six months of his life for both behavioral and physical complications resulting from opportunistic infections. Treatment of intercurrent infections permitted the patient to return to his parents' care at home. The major problems in management at home were the patient's daytime and nocturnal wanderings away from the house, inattention to personal hygiene and irregular patterns of eating, sleeping and activity. Haloperidol therapy, 2 to 4 mg given daily, helped reduce overactivity and promote regular sleep. His deteriorated judgment (smoking in bed and carelessness with home appliances and automobiles) made supervision necessary. The hospital provided a visiting nurse, and local AIDS Project volunteers supplied supervision at home on weekdays. The family reported that the information and support from the AIDS Project were invaluable, but the patient's severe amnestic state limited his ability to use the counseling services offered. Because of wasting and recurrent infections, he required hospital care during the final two months of life. The encephalitis and his central nervous system (CNS) symptoms were actively evaluated without detection of a treatable causative agent. Cerebral ventricular shunting was felt to be contraindicated because of widespread opportunistic infection. The patient ultimately died of pulmonary complications of AIDS.

An autopsy showed meningoencephalitis with severe ependymitis, ependymal necrosis and subependymal gliosis. Individual astrocytes contained both intranuclear and intracytoplasmic inclusions consistent with cytomegalovirus. The neuropathologic diagnosis was meningoencephalitis, ventriculitis and cytomegalovirus encephalitis.

Comment

Recent studies show both direct and indirect mechanisms by which AIDS can affect the central nervous system, giving rise to neuropsychiatric signs and symptoms. Indirect mechanisms thus far described include opportunistic infections (such as CMV and toxoplasmosis), primary cerebral lymphoma, secondary cerebral lymphoma, cerebral thromboembolic damage and cerebral hemorrhage.[12-14] With regard to the patient presented here, neither the cause nor the pathogenesis of the subacute encephalitis of AIDS has yet been established. CMV is a leading candidate,[12] and up to a quarter of patients dying with AIDS dementia show neuropathologic evidence of CMV infection. At the same time, neither a subcortical neuropathologic disorder nor clinical dementia correlate very well with evidence of CMV infection, indicating that this herpes virus infection may represent a secondary, superimposed process.[15] For this reason, HIV has become a prime suspect as the etiologic factor in AIDS dementia. Moreover, Ho and associates found direct HIV infection of neural and cerebrospinal fluid in 45 neuropsychiatrically affected AIDS patients.[16] In an immunologic study of serum and cerebrospinal fluid antibodies to HIV in 23 patients suffering AIDS or AIDS-related complex, cerebrospinal fluid antibodies were present in 22 of 23 cases. Of the 23 cases examined, 21 had neuropsychiatric signs and symptoms, suggesting that direct HIV infection of the central nervous system occurs in most neuropsychiatrically affected AIDS patients.[17]

Infection with HIV can lead to several medical outcomes including the development of immunity, asymptomatic immunodeficiency, autoimmune thrombocytopenia, AIDS-related complex, a wasting syndrome and AIDS.[18] Our case and those reviewed above suggest that HIV infection can also present in a psychiatrically protean fashion. Our own case showed a schizophrenia-like presentation, but affective and other behavioral disorders have also been reported. Indeed, our patient's early depressive symptoms may have represented a response to subtle cognitive impairment as well as to the burden of colitis. Although AIDS-related brain disease often appears to be the cause of such psychopathologic features, it is also recognized that AIDS may give rise to psychiatric illness as a consequence of the psychosocial stress induced by this devastating lethal illness.[19-21] While this patient showed a virulent and progressively deteriorating course, it should not be assumed that all patients with direct or indirect CNS effects of AIDS irrevocably decline. Aggressive diagnosis and treatment of CNS disease, such as toxoplasmosis or cryptococcosis, are effective, and, indeed, supportive care helped our patient to function at home until his final admission to hospital.

The ability of AIDS to mimic diverse psychiatric syndromes will require vigilance by psychiatrists and other physicians as the AIDS epidemic continues. Those persons presenting with psychiatric signs and symptoms who are identified as belonging to a high-risk group should undergo careful neurologic, neuropsychological, physical and laboratory evaluation as part of the initial and follow-up psychiatric assessments. The differential diagnosis of major psychiatric findings (psychosis, major depression) should include organic brain dysfunction as well as functional psychiatric illness. Abnormalities in neuropsychological or neuroradiologic tests may confirm the diagnosis, but an absence of positive findings should not exclude it: abnormalities on electroencephalogram, computed tomographic scan and magnetic resonance imaging are not universal, and formal neuropsychological batteries can yield equivocal results. Serial examinations may be necessary to detect underlying morphologic and cognitive disturbances responsible for the psychiatric symptoms in individual cases. The advent of promising new treatments for AIDS makes such careful assessment essential. In the future, longitudinal study of HIV-infected patients will be necessary to clarify the incidence, prevalence and natural history of psychiatric signs and symptoms in patients with AIDS.

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A 32-Year-Old Man With the Acquired Immunodeficiency Syndrome and Pneumococcal Meningitis

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THE ACQUIRED immunodeficiency syndrome (AIDS) is characterized by unusual microbiologic and neoplastic disease in patients infected with the human immunodeficiency virus (HIV).[1] Not unexpectedly, infections with these unusual microbiologic organisms may present atypically in an AIDS patient, an immunologically impaired host. In this report, a case of pneumococcal meningitis in a patient with AIDS is reported to emphasize that a common community organism, *Streptococcus pneumoniae*, may also have an atypical presentation in patients with AIDS. Prompt evaluation—but not empiric therapy—for bacterial meningitis in an AIDS patient is recommended.

(Havlir DV, Witt MD, Sande M: A 32-year-old man with the acquired immunodeficiency syndrome and pneumococcal meningitis. West J Med 1987 May; 146:618-619)

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ABBREVIATIONS USED IN TEXT

AIDS = acquired immunodeficiency syndrome
CNS = central nervous system
CSF = cerebrospinal fluid
HIV = human immunodeficiency virus
UCSF = University of California, San Francisco

Report of a Case

The patient, a 32-year-old homosexual man with AIDS, was seen because of fever and an altered mental state. Kaposi's sarcoma was diagnosed eight months before admission. Three months before admission, the patient presented with an altered mental state and was found to have cryptococcal meningitis with positive cerebrospinal fluid (CSF) cultures and a cryptococcal antigen titer of 1:512. He was treated with amphotericin B and flucytosine with improvement of his mental state and clearing of CSF cultures.

The patient lived alone and friends reported that his mental state declined during the week before admission. The patient said he did not have headaches, a stiff neck or photophobia. He reported having diarrhea for three days, with 20 bowel movements per day.

The patient had a history of syphilis, gonorrhea, *Clostridium difficile* colitis, thrush and shigellosis. His medications included ketoconazole, acetaminophen with codeine and Lomotil.* He did not smoke or drink alcohol, but he had been reported to abuse narcotics parenterally.

On physical examination he was lethargic and cachectic. His blood pressure was 98/50 mm of mercury and the pulse rate was 100 beats per minute with no orthostatic changes. His temperature was 40°C (104°F). Multiple violaceous lesions consistent with Kaposi's sarcoma were present on the skin of the face and extremities. His head showed no evidence of trauma, and his tympanic membranes were clear. He had a coated white tongue. His fundi were normal, and his neck was supple. There was no Kernig's or Brudzinski's sign. Results of respiratory tract, cardiovascular and abdominal examinations were within normal limits. The patient was oriented to name and place but not to month or year. He followed commands slowly but appropriately. The findings of the rest of the neurologic examination were nonfocal.

Laboratory values included a leukocyte count of 4,600 per μl, with a differential of 60% segmented forms, 30% band forms, 4% lymphocytes and 5% monocytes. The hematocrit was 30% (SI unit [Système International d'Unités] 0.30), serum sodium concentration was 135 mEq per liter (or mmol per liter) and potassium concentration was 2.2 mEq per liter (or mmol per liter). The value of blood urea nitrogen was 43 mg per dl (15.4 mmol per liter), serum creatinine was 2.2 mg per dl (195 μmol per liter) and glucose was 95 mg per dl (5.2 mmol per liter). The chest radiograph, electrocardiogram and urinalysis results were normal.

The patient received vigorous fluid and electrolyte replacement. He had no improvement of his mental state when given 1.6 mg of naloxone hydrochloride intravenously. A lumbar puncture was done and showed an opening pressure of 510 mm of water, with 410,000 erythrocytes and 1,400 leukocytes per μl, with 83% segmented forms, 10% lymphocytes and 7% monocytes. The CSF glucose concentration was 28 mg per dl and the protein was 1,500 mg per dl. A Gram's

*A combination product containing diphenoxylate hydrochloride and atropine sulfate.